required multiple transfusions every 3–4 weeks (altogether 27 units of packed cells) to maintain her hemoglobin at >8.0 g/dl. At term, she delivered a healthy male infant by elective cesarean section. The postpartal course and the neonatal period were uneventful.

After delivery, the patient needed no transfusion and was asymptomatic, irrespective of mild jaundice. Over the next few years, she required transfusions on only a few occasions. Five years following delivery, she underwent splenectomy for extremely increasing splenomegaly. Histologically, the spleen was compatible with congestive splenomegaly. She has since lived without any symptoms and with normal hemoglobin, as well as bilirubin levels, but she has continued to have elliptocytosis. Her child has remained healthy.

This case and those presented previously have suggested that pregnancy may precipitate severe hemolysis in a patient with HE [1–4]. Our case is unique, however, due to the huge number of transfusions required throughout the pregnancy. Furthermore, like patients with hereditary spherocytosis, hemolytic anemia due to HE improves significantly after delivery. Complete remission can, however, only be achieved by splenectomy. Our experience with HE and hereditary spherocytosis suggests that pregnancy might be associated with the increasing activity of the reticuloendothelial system, resulting in increasing hemolysis in patients with inherited defects of red cell membrane [5]. Therefore, we recommend that such patients have a splenectomy before conception.

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Acquired Central Diabetes Insipidus Complicating Acute Megakaryocytic Leukemia

To the Editor: A 46 year-old white woman presented with a 2-week history of fever, generalized bone pain, progressive dyspnea, and dizziness. The physical examination was significant for fever, pallor, hypotension, tachypnea, and tachycardia. Initial laboratory results revealed a white blood cell (WBC) count of 16,100/mm^3, with 42% blasts, a hemoglobin of 5.9 g/dl, and a platelet count of 424,000/mm^3. Initial serum sodium was normal (139 mmol/L). Bone marrow aspiration and biopsy revealed acute megakaryocytic leukemia (FAB M7). The patient became polyuric by her second day of admission (urine output = 8,820 ml/24 hr), and gradually hypernatremic (serum sodium = 158–168 mmol/L). The serum osmolarity was high (308 mOsm/kg), and the urine osmolarity was low (69 mOsm/kg). Thyroid and adrenal function tests were within normal limits. Subcutaneous DDAVP (1 μcg) was given every 12 hr. The urine output dropped to 2,458 ml over the next 24 hr, with a reciprocal increase of urine osmolarity to 368 mOsm/kg. Serum sodium and osmolarity returned to normal levels (135 mmol/L, 288 mOsm/kg) within 96 and 48 hr, respectively. All findings were consistent with the diagnosis of central diabetes insipidus. The patient died on day 12, after standard induction chemotherapy, of progressive pulmonary failure.

We report the first case of diabetes insipidus complicating acute megakaryocytic leukemia. Diabetes insipidus has been reported in other types of acute myelogenous, acute lymphocytic, chronic myelogenous, and chronic lymphocytic leukemias, as well as in myelofibrosis [1,2]. The precise mechanism for diabetes insipidus in patients with leukemia is unknown. Possible explanations include infiltration of the posterior pituitary or the supraoptic and paraventricular nuclei with leukemic cells and vascular events such as infarction or hemorrhage in these areas, as suggested by autopsy studies [2]. Monosomy 7 has been reported in several patients with AML who developed DI, but the reason for this association is unclear [3,4]. DI has been reported to resolve after chemotherapy or after BMT conditioning regimen [5].

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